by Black smokers. Poor smokers (who are disproportionately Black) could plausibly gravitate to mentholated brands because the menthol would permit more cost-effective smoking by permitting consumption of more of the total cigarette than is normally practical in the nonmenthol cigarette. In the nonmenthol cigarette, increasing irritation is perceived with continued smoking of a single cigarette because of the effects of the higher densities of tar and nicotine typically encountered in the final draws of a cigarette.

Another plausible explanation for the observed racial differences in nicotine/cotinine/thiocyanate levels not mentioned by Henningfield's editorial or by two of the papers[2,3] it cites is the increased levels of cotinine/carboxyhemoglobin in nonsmokers and smokers that may be attributed to exposure to ambient cigarette smoke at home or at work.[13,14] The higher rates of high-level serum-cotinine measures observed in Hispanics (relative to those observed in Whites) may reflect at least in part the higher frequency of smoking in Hispanic households,[15] the higher frequency of involuntary smoking in jobs held by Hispanics,[16] and the higher density of environmental tobacco smoke in the more crowded living quarters of Hispanics.[17]

In sum, current knowledge does not warrant the conclusion that was explicit in the Wagenknecht et al. article and at least implicitly supported in Henningfield's editorial and the papers he cites: that observed differences in nicotine exposure are attributable to genetically based metabolic differences. There is an absence of data concerning the impact of smoking topography on inhalation patterns and on absorption of nicotine and an absence of controls for lifestyle differences that need to be taken into account before we can begin favoring a genetic explanation for observed differences between ethnic groups in markers of nicotine exposure. □

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Pérez-Stable and Colleagues Respond

We agree with Dr. McCarthy that the topography of tobacco smoke ingestion may partially explain the higher-than-expected cotinine levels among Blacks compared to Whites. Precise measurement of the listed smoking topography variables may not be practical in conducting large epidemiological studies. Vogt and colleagues compared expired carbon monoxide and serum thiocynate to questionnaire estimates of tobacco exposure and found that questions about the depth of inhalation, the amount of each cigarette smoked, and the use of filters did not contribute significantly to the variance explained by number of cigarettes per day.[1] Their subjects were participants in the Multiple Risk Factor Intervention Trial (MRFIT) and were thus all men and mostly Whites. There is a need for additional studies with other ethnic and racial groups.

Although the observations about mentholated cigarettes are relevant, only a minority of Mexican American smokers in the Hispanic Health and Nutrition Examination Survey (HHANES) reported using a mentholated brand (18.5% of men and 21.4% of women).[2] The proportion is somewhat higher for Puerto Ricans and similar for Cuban Americans.

When cotinine is assayed using gas chromatography (as in our study[3]), the most that could be reasonably attributed to passive smoking is 0.06 μM/l (10 ng/ml) cotinine in serum. Most levels have been found to be less than 0.03 μM/l (5 ng/ml).[4] We have very limited information on environmental tobacco exposure in HHANES participants and thus cannot estimate the contribution of passive smoking to the serum cotinine levels. However, if we subtract 0.06 μM/l (10 ng/ml) from each smoker's measured serum cotinine and recalculate the cotinine/cigarette ratio, the proportion of underreporters among those smoking less than 10 cigarettes per day would decrease from 20.4 to 18.4% of men and from 24.7 to 21.6% of women. Although passive smoking may partly account for our observations, it does not invalidate our conclusions that Mexican American light smokers may underreport number of cigarettes consumed per day.

We agree that to attribute these observations of serum cotinine to genetically-based metabolic differences between ethnic or racial groups is premature and a question in need of further study. □

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Wagenknecht and Colleagues Respond

We thank Drs. McCarthy, Caskey, and Jarvik for their interest in our manuscript[1] and shall comment here on several issues. Cotinine was measured in young adult smokers in the Coronary Artery Risk Development in (Young) Adults (CARDIA) study in order to validate smoking status and quantify exposure. We agree that thiocyanate (SCN) is a poor marker of smoking status; SCN was measured in CARDIA to assess smoke inhalation in self-reported smokers and to thus provide an independent —albeit crude—assessment of comparability of self-report between the races. Consequently, we found that, within strata of the reported number of cigarettes smoked, SCN levels did not differ between races.

McCarthy et al. question the validity of this finding, given that we did not adjust for exercise or diet. With regard to exercise, CARDIA participants were asked to forgo heavy physical activity immediately prior to their morning examination, thus reducing the possible effect of exercise (diuresis) on SCN levels. Furthermore, Patterson and Block[2] have previously reported that a greater proportion of Blacks than Whites in this age group consume cruciferous vegetables (the greatest source of dietary SCN). This suggests that in Blacks SCN levels due to tobacco inhalation, are artificially high (relative to Whites), and thus, within strata of the reported number of cigarettes smoked, Black smokers may be over-reporting the number, not under-reporting, as might be expected if misreport were an explanation for the higher cigarette adjusted cotinine levels among black smokers.

We observed—as have others—the preference for Black smokers to smoke mentholated cigarettes. Ninety percent of Black smokers and 30% of White smokers report smoking mentholated cigarettes. Indeed, as we commented, such differences suggest a possible explanation for the racial differences in serum cotinine levels. However, we reported earlier that Blacks had significantly higher cotinine levels than Whites regardless of mentholation status; thus mentholation did not appear to explain the race differential. McCarthy et al. requested the alternative analysis: the effect of mentholation within race. In a multiple linear regression model similar to the one described in Table 2 of our paper, we did not observe a significant effect of mentholation on serum cotinine levels in White or in Black smokers; cotinine was 3.6 ng/ml (SE = 10.5) and 33.2 ng/ml (SE = 18.4) higher, respectively, in those smoking mentholated cigarettes. Diet, body composition, and physical fitness are unlikely modifiers of this association and were therefore not examined. The trend toward higher cotinine levels in Black smokers of mentholated cigarettes, although not statistically significant, may possibly explain part but not all of the racial difference in serum cotinine levels among smokers.

In response to the comment regarding potential racial differences in the amount of the cigarette that is smoked, we provide some yet-unpublished data. In the second follow-up examination of the CARDIA cohort (1990 to 1991), smokers and exsmokers were asked to indicate on a diagram of a cigarette how far they let their cigarette burn when they smoked. The distribution of the length that burns from smoking did not differ by race ($P > .05$). However, there was a significant racial difference in the reported amount of cigarette that burns without being smoked. (The responses were scaled on an integer scale of 1 [very little] to 4 [a great deal]). Contrary to the hypothesis of McCarthy et al., Blacks more frequently reported that a moderate amount or a great deal of their cigarette burned without their smoking it (48% vs 36%, $P < .0001$). That is, Black smokers report smoking less of their cigarette than White smokers, which is inconsistent with higher cotinine levels among Blacks.

Finally, exposure to environmental tobacco smoke is an unlikely explanation for racial differences in cotinine levels, as we have shown in our paper. Similar findings were reported by Pattishall et al.[3]; higher serum cotinine levels were observed in young Black children compared to White children after adjustment for the number of smokers in the home.

Although potential racial differences in smoking topography may provide ideas for study as noted by McCarthy et al., we were unable to explain the statistically higher cotinine levels among CARDIA Black smokers compared to White smokers by several measured differences in topography (including quantity or length of cigarette smoked and inhalation frequency) or by differences in characteristics of the cigarette (including nicotine content and mentholation). Furthermore, reporting bias and exposure to environmental tobacco smoke also do not appear to explain the observed difference.

In conclusion, we appreciate the letter of McCarthy et al. Although we find no evidence for a role of smoking topography as a cause of racial differences, we agree that these characteristics, as well as genetically based metabolic differences, should be investigated with similar vigor in order to understand the reason for higher cotinine levels among Black smokers. □

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